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Volume 14, Issue 2, March-April 2000, Pages 103-110

doi:10.1016/S0890-6238(00)00061-7 [? Cite or Link Using DOI](#)  
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**Original contributions****Lactoferrin is an estrogen responsive protein in the uterus of mice and rats**

Wendy N. Jefferson<sup>a</sup>, Elizabeth Padilla-Banks<sup>a</sup> and Retha R. Newbold <sup><sup>a</sup></sup>

<sup>a</sup> Developmental Endocrinology Section, Reproductive Toxicology Group, Laboratory of Toxicology, Environmental Toxicology Program, Division of Intramural Research, National Institute of Environmental Health Sciences, National Institute of Health, Research Triangle Park, NC 27709, USA

Received 13 October 1999; revised 24 November 1999; accepted 26 November 1999. Available online 22 May 2000.

**Abstract**

To identify lactoferrin (LF) and determine its estrogen-responsiveness in the rat uterus, immature Sprague-Dawley rats were untreated or subcutaneously injected with 17 $\beta$ -estradiol (500  $\mu$ g/kg) for 3 days and uterine tissues collected. Outbred immature CD-1 mice, treated with 17 $\beta$ -estradiol, provided the positive control. By using a polyclonal antibody raised against mouse LF, minimal detectable protein was immunolocalized in uterine epithelial cells of untreated immature rats and mice. After estrogen treatment, LF was localized in all uterine epithelial cells of both species, although staining was more intense in mice than rats. In mice, LF was evenly distributed throughout the cytoplasm with intense staining in some cells, while in rats, it was seen mainly in the apical cytoplasm. For comparison to another well-known estrogen responsive protein in rats, complement C3 was immunolocalized within epithelial cells and it showed a different staining pattern than LF. Uterine tissue homogenates were analyzed by sodium dodecyl sulfate-polyacrylamide gel electrophoresis and Western blots showed cross-reactivity with the mouse LF antibody. These findings indicate that LF is present in the rat uterus, and is induced by estrogens as reported in other species. Thus, LF is an important marker of estrogenic activity across species and will, therefore, have utility in screening for effects of environmental estrogenic compounds.

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### **Autoantibodies in the diagnosis and management of liver disease.**

**Czaja AJ, Norman GL.**

Division of Gastroenterology and Hepatology, Mayo Clinic and Mayo Foundation, Rochester, Minnesota 55905, USA. [czaja.albert@mayo.edu](mailto:czaja.albert@mayo.edu)

Autoantibodies are nonpathogenic manifestations of immune reactivity, and they may occur in acute and chronic liver diseases. Autoantibodies may be consequences rather than causes of the liver injury, and they should be regarded as diagnostic clues rather than etiologic markers. Conventional autoantibodies used in the categorization of autoimmune liver disease are antinuclear antibodies, smooth muscle antibodies, antibodies to liver/kidney microsome type 1, antimitochondrial antibodies, and atypical perinuclear anti-neutrophil cytoplasmic antibodies. Ancillary autoantibodies that enhance diagnostic specificity, have prognostic connotation, or direct treatment are antibodies to endomysium, tissue transglutaminase, histones, doubled-stranded DNA, and actin. Autoantibodies that have an emerging diagnostic and prognostic significance are antibodies to soluble liver antigen/liver pancreas, asialoglycoprotein receptor, liver cytosol type 1, and nuclear pore complex antigens. Autoantibodies of uncertain clinical value that remain under investigation are antibodies to chromatin, lactoferrin, and *Saccharomyces cerevisiae*. Continued recognition and characterization of autoantibodies should improve diagnostic precision, provide prognostic indices, and elucidate target autoantigens. These advances may in turn clarify pathogenic mechanisms, facilitate the development of animal models, and generate novel site-specific therapies.

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## Antigen specificity of circulating anti-neutrophil cytoplasmic antibodies in inflammatory bowel disease.

**Kossa K, Coulthart A, Ives CT, Pusey CD, Hodgson HJ.**

Department of Medicine, Royal Postgraduate Medical School,  
Hammersmith Hospital, London, UK.









**OBJECTIVES:** To characterize the antigen specificity of circulating anti-neutrophil cytoplasmic antibodies (ANCA) in inflammatory bowel disease (IBD). **DESIGN:** Analysis of the prevalence of circulating ANCA in patients with ulcerative colitis and Crohn's disease, by both non-specific methods (immunofluorescence against fixed neutrophil leukocytes) and specific antigen techniques (against purified neutrophil leukocyte constituents). **METHODS:** Indirect immunofluorescence against fixed polymorphonuclear leukocytes, and solid-phase enzyme-linked immunosorbent assay (ELISA) against neutrophil constituents (alpha-granules, elastase, myeloperoxidase, cathepsin g, lysozyme and lactoferrin). **RESULTS:** Although results using immunofluorescence were typical of other studies (ulcerative colitis positive in 41%, Crohn's disease in 10%), ELISA studies showed antibody activity against neutrophil components in 69% of patients with ulcerative colitis and 39% of those with Crohn's disease. Antibodies in ulcerative colitis were commonly directed (in descending order) against lysozyme, cathepsin G, elastase, and lactoferrin, and in Crohn's disease against lysozyme. **CONCLUSION:** Correlation of indirect immunofluorescence data and ELISA results indicated that even this large panel of specific antigens fails to identify all the ANCA targets in IBD. The lack of correlation between the findings of ANCA, either in general or versus a specific target, and disease extent or activity in ulcerative colitis supports the suggestion that ANCA are unlikely to be of primary importance in pathogenesis.







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